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## ORIGINAL ARTICLES.

# ACUTE DILATATION OF THE STOMACH, AND ITS RELATION TO MESENTERIC OBSTRUCTION OF THE DUODENUM.

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Although in recent years contributions to the subject of acute dilatation of the stomach have been fairly numerous in England and Germany, in this country the subject has received but little attention. Nevertheless the condition is one of considerable clinical interest and importance. Cases are encountered by the surgeon, the gynecologist and the orthopedist, as well as by the physician, and the condition has developed during apparently normal convalescence from operations or from some severe illness, changing, within a few hours, perhaps, the whole aspect of the case. While in most recorded instances the disease has progressed rapidly to a fatal termination, evidence is accumulating to show that in many cases life may be saved by timely recognition of the condition and by suitable treatment.

The possibility of such acute dilatation has long been known. Brinton, writing in 1859, discusses the subject and gives a short account of its clinical features. It was Hilton Fagge, however, who first directed general attention to the condition and gave an accurate description of the characteristic symptoms and physical signs. Since that time (1873) cases have gradually accumulated, until in 1902 Campbell Thomson was able to collect 44 instances of the disease—most of them with autopsy reports. The more recent review of the subject by Neck was based upon 60 cases. For the present study of the condition 102 cases have been collected and analyzed.

The accumulation of cases has made it evident that, in many instances at least, the clinical picture is very distinct and easily recognizable, and that the diagnosis is often not difficult if the possibility of the disease is borne in mind. There is also reason to believe that with the earlier recognition of the condition it will be possible to treat successfully many cases which have heretofore proved fatal. Concerning the pathogenesis of the disease, however, our knowledge is by no means so satisfactory and there exist still much confusion and uncertainty as to the nature of the affection and its causation.

In studying the recorded cases it soon becomes apparent that the condition of acute dilatation of the stomach rests upon no single pathological basis. A consideration of the extremely diverse etiological features and of the varying postmortem findings makes this very evident. Nevertheless it seems to have been clearly demonstrated within the last few years that a very important cause—perhaps the most important single cause—of such stomach dilatation is the sudden incarceration of the lower end of the duodenum between the root of the mesentery which passes in front of it and the vertebral column behind. The possibility of such mesenteric obstruction of the duodenum was expressly noted by Rokitansky in 1863. No illustrative cases were recorded, however, until 1891, when Kundrat, under the caption "Concerning a Rare Form of Intestinal Incarceration," described three such cases. These all had run a rapidly fatal course, and in two of them the necropsy showed great dilatation of the stomach as well as of the duodenum. Schnitzler, in 1895, reported another fatal case with enormous dilatation of the stomach and duodenum, and also an apparently similar case cured by treatment directed to the relief of the supposed duodenal compression.

The subject received no other notice, however, until Albrecht, in 1899, published a very complete and careful study of it. He reported two cases of acute gastric dilatation with the same mesenteric constriction of the duodenum, and discussed at length the relation of the two conditions to each other. Since Albrecht's communication a good deal further study has been made, chiefly in Germany, and a number of additional cases have been recorded. It should be mentioned that in 1900 Robinson, of Chicago, without knowledge of the literature of the subject, and believing that he was describing a hitherto unrecognized condition called attention to the possibility of such compression of the duodenum by the root of the mesentery and reported a fatal case associated, as usual, with enormous dilatation of the stomach.

The following similar case occurred in my service at the New York Hospital, and through the kindness of Dr. Farr of the house staff I was fortunate enough to secure photographs of the postmortem appearances.

Male, aged twenty-one years, Italian tailor, was admitted to the hospital on August 1, 1905. He had had malaria ten years before in Italy, but otherwise had always been perfectly well except for an occasional slight cough. There was no history of tuberculosis in the family. For four weeks before admission he had noticed progressive weakness and loss of flesh, had had slight cough, some dyspnœa, and some profuse night sweats. No pain. During the last few days he had been irrational at times. The bowels were constipated. Admission: temperature, 99.8°; respiration, 36; pulse, 116.

*Physical Examination.* Man of medium frame, very poorly nourished; mucous membranes pale. Pupils of equal size, reaction normal. Tongue moderately coated. Throat congested and dry. Neck somewhat stiff. No subcutaneous œdema. Lymph nodes in neck, axillæ, and groins palpable.

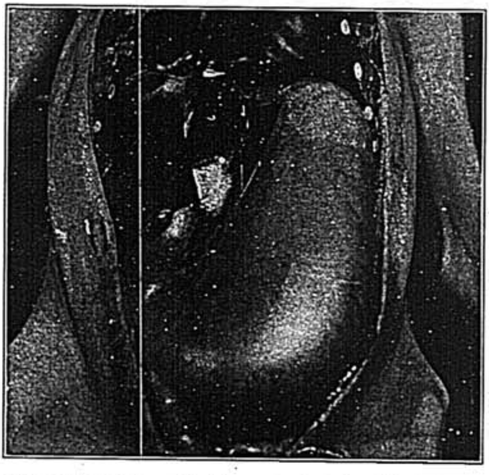

FIG. 1.—The dilated stomach due to mesenteric obstruction of the duodenum.

*Lungs.* In front slight dulness at right apex. Exaggerated vocal fremitus over same area. Vesicular breathing everywhere. Sibilant and sonorous rhonchi over both chests. Some medium-sized moist rales, especially with expiration, in both axillæ. Behind, slightly impaired resonance at the right apex. Auscultation signs similar to those in front.

*Heart.* Maximum impulse in fourth space four and one-half inches to left of midsternal line; action feeble, regular, and rapid. Relative dulness begins above at third rib, on the right side at right border of sternum, on the left side at line of apex. Sounds all normal except some accentuation of second pulmonic. Pulse rapid. regular, small, and weak. Artery walls somewhat thickened.

*Abdomen.* Retracted and somewhat rigid, has rather a doughy feel, but no masses can be felt. No rose spots. Liver dulness reaches to two fingers' breadth below costal margin, edge indistinctly felt. Right kidney palpable. Edge of spleen felt at costal margin.

The temperature rose at once to 103° and thereafter ranged constantly between 100° and 104°, with morning remission and evening rise, forming a chart very suggestive of typhoid fever.

Repeated examinations of the sputum throughout the illness yielded negative results for tubercle bacilli. Tests for the Widal reaction were all negative. Leukocytes: August 2, 34,000 in c.mm.; 8th, 26,000; 21st, 12,000.

*August* 15. Hemoglobin, 56 per cent.; red cells, 4,000,000. Examination for malarial plasmodia negative. Blood cultures taken on two occasions failed to yield any growth. Urine showed at each examination a trace of albumin and a few hyaline and granular casts. Diazo reaction positive.

*5th.* Condition and physical signs almost unchanged since his admission. Fever continuous. Mental state "queer." Takes very little food.

*9th.* Small moist rales scattered irregularly throughout both chests anteriorly; edge of spleen now felt over finger breadth below costal margin.

*14th.* Still slight dulness at right apex in front. Auscultatory signs of general bronchitis.

*23d.* General condition worse. Involuntary micturition and defecation. Will take almost no food, but drinks water freely. Fed by gavage. Spleen is decidedly larger than on admission. Complains of no pain. Belly retracted and somewhat rigid. No rose spots. Diagnosis of acute miliary tuberculosis seems very probable by exclusion, but in the absence of evidence of ocal tuberculosis cannot be made with certainty.

*28th.* Patient has been eating better for past day or two. Slept soundly last night. This morning about 9.30 suddenly complained of severe abdominal pain. He was seen almost immediately by the house physician, who found a marked distention of the left side of abdomen, especially above the umbilicus. Lower border of this circumscribed prominence could be made out just below umbilicus. The distended area gave deep tympany on percussion, was very tender to pressure, but there was no real rigidity of the abdominal muscles. The severe pain continued, the general condition grew rapidly worse, and at 12.40 P.M. (three hours after onset of pain) he died. There was no vomiting at any time.

*Autopsy.* Two hours after death (Dr. Biggs).

*Anatomical Diagnosis.* Acute general miliary tuberculosis; chronic tuberculosis of the bronchial and mediastinal lymph nodes; enormous dilatation of the stomach; obstruction of the duodenum by the root of the mesentery.

Body greatly emaciated, slight rigor mortis. No subcutaneous œdema. Abdomen somewhat distended. Upon opening the abdomen an enormously enlarged stomach presents. Peritoneum normal. The pericardial sac contains 150 c.c. of clear serous fluid. Near the base of the right ventricle is a miliary tubercle in the visceral layer. The heart is small, the cavities slightly dilated, the muscle pale and soft, the valves and coronary arteries normal. The bronchi contain much mucopus. The lungs are of light

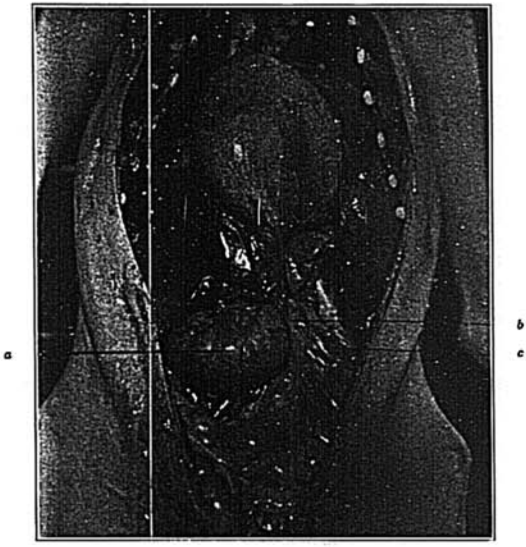

FIG. 2.—View of same case, after the stomach had been turned upward and the transverse colon removed, showing the greatly distended duodenum, the constricting root of the mesentery, and the collapsed first portion of the jejunum. *a.* Dilated duodenum. *b.* Root of the mesentery. *c.* Collapsed jejunum.

color and very emphysematous. Miliary tubercles are numerous beneath the pleura and throughout both lungs. No old tuberculous lesions are found in any part of the lungs. The bronchial and mediastinal lymph nodes are much enlarged and show extensive caseation, but no softening. These caseous nodes extend up along and behind trachea into neck. The spleen is greatly enlarged; it measures 17.5 by 11.5 by 8 cm., and weighs 620 grams. The great size is due to presence of myriads of tubercles, 2 to 4 mm. in diameter. Remaining splenic pulp very hyperemic, so that tubercles stand out sharply. The left kidney weighs 140 grams, the right 185 grams. Few small tubercles in each; capsules not adherent; surface smooth; markings distinct; pelvis and ureters normal. The stomach is enormously enlarged and distended by gas and 650 c.c. of greenish-brown fluid. Greater curvature extends to within 7 cm. of symphysis. Distance from fundus to lowest part of greater curvature is 31 cm. Pylorus in normal position and patulous. Duodenum 7 cm. in diameter and tensely distended with fluid to point where mesentery crosses it. Remainder of small intestine is collapsed and lies chiefly in pelvis. Owing to catarrhal inflammation, slight œdema and absence of gas; the small intestine is heavy and with its mesentery weighs 750 grams. The weight of this mass as it hangs into the pelvis drags heavily on the mesentery and causes firm pressure upon the terminal part of duodenum. A finger introduced into duodenum encounters complete obstruction at this point, which is relieved by raising the mesentery. In this constricting band of mesentery the superior mesenteric artery can be felt as a tense, firm cord. No structural changes in duodenum or pylorus. The stomach wall is very thin. Pancreas normal. The suprarenal bodies are normal except for a single tubercle in the right one. Liver weighs 1600 grams, and is normal except for the presence of vast numbers of tubercles. Lymph nodes about pancreas, lesser omentum, retroperitoneal region, groin, and axillæ are all somewhat enlarged and most of them contain fairly large tubercles. Mesenteric nodes slightly swollen. No tubercles. The other organs are normal.

Microscopically the stomach mucosa shows a fairly marked catarrhal inflammation. The muscular layer seems about half its usual thickness. There is no apparent separation of the muscle bundles, and no structural change appreciable in the fibers.

This case differs from most of those reported (1) in the remarkably short duration of the symptoms, and (2) in the entire absence of vomiting. No immediate cause for the development of the condition could be discovered. The patient had for many days taken only very small quantities of liquid food and that morning had had only a part of a glass of milk. The bowels had moved almost daily up to the time of his death. While there is no certainty that the stomach may not have been dilated before his terminal symptoms began, there had been nothing to suggest this. He had had no gastric symptoms other than anorexia, and, although his abdomen had been examined frequently and carefully, there had been found no increase of stomach tympany, no splashing, no visible peristalsis. During the last week or two of his life he had kept the dorsal position in bed almost constantly. The probable influence of this, and of the almost complete abstention from food, in favoring the production of mesenteric obstruction of the duodenum will be discussed later.

In addition to the case just described I am able, through the courtesy of Dr. James Ewing, to furnish brief notes of another instance of acute dilatation of the stomach.

The patient, a man aged twenty years, was under observation for about six weeks and presented the picture of chronic pulmonary tuberculosis, with hectic fever, chills, emaciation, anorexia, and cough. Aside from occasional vomiting with the paroxysms of coughing, he presented neither symptoms nor physical signs of any stomach disorder. His condition remained practically unchanged until two days before his death, when he suddenly began to vomit large quantities of greenish fluid. His urine became scanty and bloody, vomiting persisted, his condition changed rapidly for the worse, and he died within forty-eight hours of the onset of the violent gastric symptoms.

The autopsy revealed, in addition to the lesions of advanced pulmonary tuberculosis, a stomach enormously distended and reaching to within three inches of the symphysis pubis. No lesion was found to explain the enormous dilatation. The small intestine showed no abnormality other than a moderate grade of congestion. There was no peritonitis.

ETIOLOGY. The following study of acute dilatation of the stomach is based upon an analysis of 102 cases:

*Age.* In 90 cases in which the age of the patient is given the percentages according to decades are as follows: First, 4 per cent.; second, 20 per cent.; third, 36 per cent.: fourth, 19 per cent.; fifth, 14 per cent.; sixth, 5 per cent.; seventh, 2 per cent. It will be noted that three-fourths of all the cases developed during adolescence or in early adult life, *i. e.*, between the tenth and the fortieth years. The youngest case was that of a child of three years (Dickinson); the oldest that of a woman of sixty-nine (Kundrat).

*Sex.* Males were affected in 47 per cent. of the cases; females in 53 per cent.

In studying the state of health of the patients at the time of the onset of the symptoms, and the conditions which seemed to bear on the orgin of the disease, it is seen that the cases fall into several more or less well-defined groups:

1. *Those following Operation with General Anesthesia.* These constitute much the largest group and include 42 cases (41 per

cent.); 15 followed various operations upon the gall-bladder and bile passages (Riedel, Robson and Moynihan, Kehr, and others); 17 developed after other abdominal operations, such as removal of the uterus (v. Herff, Braun), operations on the kidney (Morris, Thomson, Borchardt), etc.; and the remaining 10 cases after operations upon various other parts of the body, chiefly joints. Thus the condition has complicated excision of the knee (Goodhart); of the hip (Jessop); of the elbow (Albrecht); amputation of the thigh (Box and Wallace, Goodhart); amputation of the breast (Albrecht); tenotomy of the tendo Achillis (Müller), etc. It is worthy of note that no instance was found in which acute dilatation had followed operation upon the stomach itself. In almost all the cases in which the nature of the anesthetic used is mentioned it has been chloroform. In one instance, however (Herrick), it is noted that ether (preceded by nitrous oxide gas) was used; and in a number of other cases in which the anesthetic is not mentioned it seems altogether probable that ether was the drug employed.

The time of the advent of the symptoms of dilatation could be determined with reasonable accuracy in 32 cases. In 16 of these they appeared within the first twenty-four hours after operation, and the usual postnarcotic vomiting merged imperceptibly into that of the acute dilatation; in 7 cases the symptoms appeared during the second twenty-four hours, and in 7 others between the third and the seventh day after operation. Once (Robson, Case I) the condition manifested itself, without known cause, on the tenth day of convalescence, and again (Robson, Case III) two weeks after operation. In this latter case the symptoms appeared after eating a raw apple. In a number of the cases the stomach had been seen at the time of operation and nothing abnormal had been noted.

2. *Those Occurring in the Course of or during Convalescence from Severe and Wasting Diseases.* The condition has been met with five times in the course of lobar pneumonia, three times in chronic pulmonary tuberculosis, and once each in general miliary tuberculosis, long-standing hip-joint disease, severe and protracted sciatica, severe chorea, grave anemia, and inoperable sarcoma of the humerus. Meyer has seen it develop in a case of mental disease of long standing (without polyphagia). The symptoms have appeared during convalescence from typhoid fever in four instances. In one of these (Kundrat) they developed immediately upon the patient leaving his bed for the first time. The condition has also been seen during convalescence from severe scarlet fever, from pneumonia, and from some obscure fever. In this group the features common to most of the cases have been long-continued decubitus, prostration, and emaciation.

3. *Cases Developing after Injuries.* Acute dilatation of the stomach has followed injury to the head or spine in four instances.

Rosenheim observed it after a fall upon the shoulder and head (without fracture). In Campbell Thomson's patient it followed fracture of the base of the skull and of the cervical spine. Edmunds records a case of shot wound of the lower dorsal spine, with complete paraplegia, in which thirty-four days after the injury symptoms of acute dilatation suddenly appeared and terminated fatally in nine hours. Erdmann saw the immediate development of dilatation after a fall upon the back in which the body was bent sharply backward. A slight blow upon the abdomen seemed to be the exciting agent in a case of Box and Wallace.

4. *Cases Ascribed to Errors in Diet (Dilatatio ex Ingestis).* Indiscretion in diet is given as the direct or contributing cause in fifteen instances; with what justification it is not always easy to determine. Nine times the symptoms developed after a single large meal in persons in good health (Boas, Broadbent, Fränkel, Heine, Hoffmann, Körte, Kirch, Kundrat, and Valsalva). In Broadbent's case the patient had been tramping for two days without food. After a large meal of roast pork he developed severe abdominal pain and vomiting and died at the end of two days. In Heine's case the stomach had been examined a short time before and had been found to be normal in size and in motor function. The symptoms began in Kirch's patient after a supper of two plates of soup and a glass of beer. Six times other possible causes existed along with errors in diet. In Bennett's patient, who was ill of empyema, the symptoms followed the drinking of two bottles of effervescent lemonade. In two instances (Buzzard, Kelling) the patients were encased in plaster jackets (for spinal deformity), and the symptoms appeared after a copious meal. Acute dilatation developed in a woman seven months pregnant after bodily fatigue and the drinking of a glass of cold milk (Dyson). In a case of valvular heart disease dilatation and death supervened after distention of the stomach with food (Goodhart). A patient convalescent from typhoid fever developed evidences of dilatation of the stomach after drinking a very large amount of fluid (Heine).

5. *An Interesting Group of Cases is that Associated with Disease and Deformity of the Spine.* There are 6 such cases; 3 of these (Buzzard,[1] Kausch, 2 cases) were instances of Pott's disease with dorsal kyphosis and (in 2 cases at least) increased lumbar lordosis. In 1 case (Willett) there was a uniform posterior (kyphotic) curve of the whole dorsal and lumbar regions; in another (Kelling) a dorsolumbar scoliosis, and in a third (Kirch) a rachitic dorsal kyphoscoliosis. A further interesting fact is that in 3 of the above cases the gastric symptoms became manifest while the patients were enveloped in a plaster-of-Paris corset (Buzzard, Kelling, Willett).

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[1] This case is often credited to Perry and Shaw.

6. Finally there are 4 cases (W. H. Brown, Miller and Humby, Simon, Wright) in which the symptoms of acute stomach dilatation appeared while the patients were in their usual good health, and for which no cause whatever is assignable. In another case (Schmorl) the symptoms appeared immediately after a violent paroxysm of laughing.

SYMPTOMS. Vomiting, pain, abdominal distention and tenderness, constipation, thirst, scanty urine, and rapid collapse are the chief and common symptoms.

*Vomiting* is unquestionably the most constant and most important. It has been present in over 90 per cent. of the cases reported. In only 2 cases (v. Herff, Case II; Conner) has its absence throughout been specifically noted, although in 3 other instances (Borchardt, Dickinson, Edmunds) no mention is made of it among the symptoms. The vomiting in most cases appears at the beginning and persists throughout the illness. In a number of instances, however, it has been noted that the vomiting ceased a day or two before death, and once (Bennett) it began only after severe abdominal pain had lasted for five days. "Persistent," "incessant," "uncontrollable," "profuse," are the terms usually used in describing it. The quantity of fluid vomited has varied greatly, but in general it has been noticeably large. Many writers comment upon the surprisingly large amounts brought up. Morris was so struck with the quantity vomited in his case that he looked upon the excessive secretion of fluid as the primary condition and the dilatation of the stomach only as the result, and he proposed for the condition the name of "acute gastrorrhœa." In Miller and Humby's case "five basinfuls" of fluid were vomited during one night, and in a number of the cases as much as several quarts of fluid have been removed from the stomach at one time.

The vomited material is usually of thin, watery consistence. Its color varies much, but is generally some shade of green. Occasionally it is "brown" or "blackish," and in many instances the vomit is described only as "bilious." There can be no doubt that bile is present in the vomit in most of the cases. In several instances small quantities of blood could be detected in the stomach contents. A feature of much diagnostic importance is the fact that although the vomitus has sometimes been foul-smelling, it has only rarely been feculent. In 3 instances only was the vomit noted as distinctly fecal (Kundrat, Lehmann, MacEvitt), and in 1 other (Willitt) it is described as "stinking."

Concerning the chemical character of the fluid vomited or removed by tube but little has been learned. In both of Baümler's cases an absence of free HCl was noted. In one of these lactic acid was present and the fluid showed also the biuret reaction and the presence of a diastatic ferment. Herrick found bile present, a total acidity of 18 units, no free HCl, and no lactic acid. Kelling's case showed no free HCl, but much lactic acid. On the other hand, Heine found a total acidity of 48 units, the presence of free HCl, and an absence of lactic acid. The presence of hydrogen sulphide was noted by Boas and Heine.

*Pain.* Definite statements as to the presence or absence of abdominal pain are found in only 42 cases. In these pain of greater or less severity was present 35 times. In the remaining 7 cases no pain occurred. The pain was referred usually to the epigastrium or the umbilical region, and in many of the cases was described as very intense.

*Tenderness.* The presence or absence of tenderness is recorded but 17 times. It was present in 12 and absent in 5 of these cases.

*Distention* was present in most cases. Its absence was noted only 4 times. It affected chiefly the epigastrium and the region to the left of the umbilicus, but in a few instances it is described as having been almost altogether below the navel. In others it was general and globular. Fagge describes the distention as lying below a line running obliquely downward and to the right to the right iliac region.

In a few instances *muscular rigidity* was noted, but oftener its absence is recorded.

*Constipation.* In many of the reports no record of the state of the bowels can be found. In 14 cases, however, there was no passage either of gas or feces. In 9, on the other hand, diarrhœa existed. Often the symptoms are described only as being those of intestinal obstruction.

The *temperature,* except in cases in which the symptoms have developed in the course of febrile diseases, has been almost invariably normal or subnormal. In a few of the fatal cases there has been a moderate rise of temperature toward the end.

*Thirst* is described as intense in many of the cases, as might be expected, and complete suppression of, or great diminution in the amount of, the urine is frequently noted. This is not an invariable rule, however, for in 2 instances with persistent vomiting it is expressly stated that no diminution in the quantity of urine occurred. Indican was present in the urine in some cases.

*Persistent hiccough* was noticed in a few of the patients, as was also a terminal delirium. Severe muscular cramps throughout the body occurred in 1 case (Wright), but no instance of tetany is recorded.

*Splashing sounds* and fluctuation waves below the level of the umbilicus aided in the diagnosis in a considerable number of the cases.

*Visible Peristaltic Waves.* A notable feature of the physical signs has been the absence of visible peristalsis. Only 3 exceptions to this rule could be found. Baümler and Herrick each saw slight peristaltic waves over the stomach. Schultz also saw slight waves on one day, but on the next, when the dilatation had increased, they had disappeared.

Next to the vomiting the rapid development of *collapse* is perhaps the most striking feature of the clinical picture. The sunken eyes, the pinched and livid features, the superficial breathing, and the small, empty, very rapid pulse suggest at once some grave abdominal lesion and serve to explain the common diagnosis of perforative peritonitis or intestinal obstruction.

DURATION AND PROGNOSIS. Among the 102 cases collected there were 74 deaths (72.5 per cent.) and 28 recoveries. It seems very unlikely, however, that this represents the true ratio between the patients that die and those that recover. There are probably many instances of moderate grade of acute stomach dilatation which get well without being recognized or, at least, without being recorded. In 63 of the fatal cases the duration of the symptoms could be determined with reasonable accuracy, and was as follows: Less than twenty-four hours, 7 cases; one to two days, 11 cases; two to three days, 14 cases; three to five days, 16 cases; five to ten days, 7 cases; ten to sixteen days, 8 cases. In other words, the duration of the symptoms in 75 per cent. of the fatal cases was less than five days. The 2 shortest cases were that of Dickinson (three or four hours) and my own (three hours); the longest that of Kausch (sixteen days). In the patients that recovered the duration of the symptoms varied greatly. In a few postoperative cases these disappeared almost immediately upon change of posture or some other therapeutic measure, but in other instances the dilatation subsided only very gradually (in the course of weeks), and once, at least (v. Herff), a moderate degree of dilatation persisted.

MORBID ANATOMY. The statements which follow are based upon an analysis of the records of 69 autopsies.

The most noteworthy feature of the postmortem picture is the enormous size of the stomach. In almost every autopsy protocol the extreme degree of dilatation is commented upon. Often the organ occupies almost the whole of the abdomen. In 1 instance it is described as reaching into the true pelvis. In a number of cases it extended to the symphysis pubis. Once at operation it was mistaken for an immense pancreatic cyst, and as such was evacuated and drained (W. H. Brown). Riedel describes its color as seen at operation as purplish red. In several instances, however, it was grayish or bluish white (Robinson, Fenger, MacEvitt). The distended stomach assumes in most cases a characteristic cylindrical form and is sharply bent into two unequal portions. The larger of these—the cardiac portion—extends almost perpendicularly downward, while the smaller and shorter pyloric limb extends upward and to the right, producing a sharp, angular bend in the lesser curvature and giving to the organ a somewhat V- or U-shape.

Among the whole 69 autopsies definite obstruction at the pylorus was found only three times: once by tumor (Thomson, Case I), once by constriction by a band of mesentery (Riedel, Case III), and once by kinking from adhesions (Riedel, Case II).

The stomach wall is described as abnormally thin in 14 cases. In 3 instances (Albrecht, Case II; Müller, Cases I and II) the wall is mentioned as of normal thickness. In 1 case (Kundrat, Case III) it is described as "thick." In none of the other cases is the condition of the wall mentioned.

A histological examination of the stomach wall is recorded in only a small proportion of the cases. In 1 (Schultz) there were found hemorrhages into the muscular layer, which seemed much thinner than normal and in which the muscle bundles were in many places torn apart and widely separated. In Miller and Humby's case also the muscle fibers were found torn apart. In the few remaining cases, including my own, in which a microscopic examination was made, the musculature presented no appreciable change other than the thinning. The wall in several cases showed many minute hemorrhages in the mucous and submucous layers. In 2 cases (Stieda; Fraenkel, Case II) the mucosa was the seat of small superficial erosions. In 1 instance (Körte) there existed an area of necrosis in the fundus, with perforation of the wall. In a very few cases there were present the lesions of chronic gastritis.

*Duodenum.* A part or the whole of the duodenum was found dilated in 38 cases (55 per cent.). In almost all of the remaining ones no mention whatever is made of the condition of this part of the intestine, and, as in many of these the autopsy records are very scanty and incomplete, it appears altogether probable that in some of them a dilatation of the duodenum may have been overlooked or, if noticed, not recorded. It seems, therefore, safe to say that in quite two-thirds of the cases, at least, the dilatation is not confined to the stomach, but involves as well a part or the whole of the duodenum.

In 19 of the 38 cases in which the duodenum was dilated the dilatation stopped suddenly near its lower end, at the point where the duodenum passes behind the root of the mesentery, and definite obstruction existed here by the pinching of the gut between the tense mesentery and its contained superior mesenteric artery in front and the aorta and vertebral column behind. That the pinching of the duodenum by the tense mesenteric band is real and serious is abundantly proved by the autopsy findings in Baümler's case, which revealed, in addition to a greatly dilated stomach, a dilatation of the duodenum to the point where it was crossed by the root of the mesentery. At this place the gut was compressed and showed a sharply defined band of bright-red color 2 cm. wide, and corresponding to this ring a necrosis in the mucosa 1.5 cm. wide. Microscopically this band of necrosis was found to be deepest at its centre, where it reached almost to the submucous layer.

In 8 other cases the dilatation ceased at the lower end of the duodenum, but compression by the mesentery was not noticed. These cases are those of Box and Wallace (Cases II and III), Buzzard, Fenger, Meyer, Petit, Schultz, and Thomson (Case V). In Buzzard's and Petit's cases the dilatation was ascribed to a sharp kink at the duodenojejunal junction. In Schultz's case, in which the duodenum had a diameter of 9 cm., the compression at the lower end was thought to result from the weight of the greatly dilated stomach. Meyer suggests the same explanation in his case. It seems very probable from the descriptions that some of these 8 cases may have been instances of mesenteric obstruction.

Three times (Hood, Jessop, Kirch) the dilatation involved not only the duodenum, but the upper few inches of the jejunum as well. In 1 instance (Mahomed) the duodenum and "intestines generally" were dilated. In another case (Kausch, Case I) the first portion (superior limb) of the duodenum was enormously dilated, and at the junction with the descending portion was kinked at an angle of 180 degrees with this latter. In Balster's patient there was an extravasation of blood (of unknown cause) in the wall of the descending limb, which caused constriction of the lumen, with great dilatation above.

The state of the intestines below the duodenum is not noted in many cases; when it is mentioned, however, they are described as empty and collapsed. In only 1 instance (Mahomed) are they spoken of as distended. In almost every case of mesenteric obstruction the small intestine has been found collapsed and lying chiefly in the true pelvis.

THE MECHANISM OF ACUTE DILATATION OF THE STOMACH. From the time of Brinton this condition has been looked upon by most writers as primarily of paralytic nature. The absence in almost all cases of obvious obstruction at the pylorus, and the fact that a similar paralytic distention of the intestines is a not uncommon phenomenon after abdominal operations and in peritonitis, have served to support this view; as has also the experimental work of Carion and Hallion. In practising, for other purposes, section of the vagi at varying levels, these investigators noted that gastrectasis followed the operation. "After resection of both nerves above the diaphragm we have seen produced in the dog an enormous dilatation of the stomach and of that portion of the œsophagus innervated"—and they add: "If section of the vagus nerves is capable of engendering complete atony and permanent dilatation of the stomach, it is hardly disputable that effects of the same nature could be determined by paralysis or inhibition of the corresponding nervous centres without any previous alteration in the stomach itself."

Accepting the usual teaching that the vagi supply the motor impulses to the stomach musculature, while sympathetic fibers from the dorsal cord, through the solar plexus and splanchnic nerves, carry inhibitory impulses to control such peristaltic movements, the 2 cases (mentioned under Etiology) in which injury to the head has been followed by acute gastric dilatation would seem to be illustrations of such central paralysis of the motor function; and the cases following injury to the dorsal spine might perhaps be looked upon as due to abnormal stimulation of the inhibitory nerves. In the same way the 5 cases which developed in the course of severe pneumonia (Box and Wallace, Herrick, Hood, Lehmann, Thomson) might, it is conceivable, result from the involvement of the vagus trunks in the inflammatory exudate of the posterior mediastinum. It happens that in 4 of the 5 cases there was involvement of both lower lobes, and in the fifth case consolidation of the right lower lobe, with extensive plastic pleurisy over the bases of both lungs—in other words, in all 5 cases the conditions were favorable for the extension of the inflammation to the neighborhood of the vagi. Unfortunately in none of the postmortem examinations of these cases was the question of the involvement of the vagi investigated.

While in a few of the cases it is possible, then, that the explanation of the sudden dilatation may lie in some disturbance of the nerve centres or nerve trunks, this probably does not hold good for most cases. In the great majority of instances the disturbing agent must act directly upon the stomach musculature or its terminal nerve filaments. It has been suggested that the excretion of chloroform by the stomach may explain the large class of postoperative cases (v. Herff). Albu in his case ascribed the paralysis to the absorption of toxins formed by abnormal fermentative processes in the stomach. In other instances blows over the stomach, mechanical injury during operation, local peritonitis, as well as overdistention, severe infections, rapid emaciation, etc., have been regarded as the cause of such paralysis.

The view that acute dilatation depends upon a primary paralysis of the stomach musculature has not been universally held, however. Robson concludes that an analysis of all the cases "would seem to point to a neuroparesis associated with spasm of the pylorus, and Pepper and Stengel also believed some of the cases, at least, to be obstructive, "the pyloric stenosis being due to spasm, the result of acute indigestion or hyperacid secretion."

The theory of pyloric spasm as an explanation for any considerable proportion of the cases seems untenable, in view of the facts that in most of the cases the vomitus contains bile and that in about two-thirds of the cases the dilatation involves also the duodenum.

Morris was disposed to look upon the excessive secretion of fluid ("acute gastrorrhœa") as the primary condition and the dilatation as secondary, but there seems to be little basis for such a view.

With the accumulation of cases of acute gastric dilatation associated with mesenteric constriction of the duodenum, the question has very naturally arisen whether most cases of dilatation might not be due to such obstruction of the duodenum. That many instances of dilatation have not shown this obstruction postmortem would not in itself disprove such a theory, since in the usual manipulation of the intestinal coils the condition might very easily be overlooked unless the possibility of its occurrence were borne in mind.

The whole subject of the mechanism of acute gastric dilatation has been carefully studied by Kelling, and while one may hesitate to accept all his conclusions, his work seems to throw some light upon a problem which at best is complex and puzzling. Kelling starts out with the assumption that, as a prerequisite to the production of acute dilatation, there must be some obstruction to the exit of the stomach contents (fluid and gaseous) *both* upward and downward, for otherwise the overdistention of the organ (by which alone, he thinks, acute dilatation can occur) will be prevented by vomiting and eructation or by the passage of the stomach contents into the intestines. In a series of experiments upon cadavers he found, by inflating the stomach through an opening in its anterior wall, that in a considerable proportion of cases the cardiac orifice was impervious to the passage of air from the stomach into the œsophagus no matter how great the pressure might be; in other cases air would pass only when the intragastric pressure was high, and in still others it passed so freely into the œsophagus that no considerable intragastric pressure could be secured. He found this valve-like closure of the cardia to depend upon certain anatomical relations between the fundus of the stomach and the insertion of the œsophagus. In such cases the cardia can be opened (from below) only by active contraction of bands of muscle fibers running from the œsophagus into the wall of the fundus, and if, by a weakening of these muscles from any cause or by a great increase in intragastric pressure, the active opening force fall below the pressure which maintains the closure of the cardia, any emptying of the stomach through the œsophagus is rendered impossible.

With the œsophagus securely closed by ligature Kelling found, further, by inflating the stomach, that in certain of the cases air passed freely through the duodenum, but that in a majority of instances obstruction occurred by the formation of a sharp kink in some part of the duodenum. Sometimes this kink formed at the junction of the superior with the descending limb, at the point of attachment of the ligamentum heptoduodenale, and above the papilla of Vater; in other cases it occurred below the papilla—usually at the junction of the descending limb with the inferior horizontal portion. The production of such kinks (the possible occurrence of which has long been recognized) seems to depend upon the firmness with which the duodenum is attached to the posterior abdominal wall. In cases in which the gut is relatively loosely fixed it is able to alter its position sufficiently to prevent such kinking.

It is those individuals, Kelling thinks, who have the tendency both to valvular closure of the cardia and to the production of an obstructive kink in the duodenum who are liable, with overdistention of the stomach by gas or food, to the development of acute dilatation.

I have myself repeated Kelling's postmortem experiments in a series of 12 cases (adults dying of affections not involving the stomach or duodenum) with results which differ somewhat from those he obtained. In 7 instances very considerable distention of the stomach occurred before there was any leakage of air through the œsophagus, but such leakage always took place when the air pressure reached a certain height, which, measured by a water manometer, varied between 15 and 26 cm. In the other 5 cases air passed freely into the œsophagus and no distention of the stomach could be produced.

My results in the matter of duodenal obstruction were also somewhat at variance with those of Kelling. With the œsophagus ligated and the stomach inflated, obstructive duodenal kinks were seen in only 3 cases, and in each of these air passed the obstruction when the intragastric pressure measured 22 cm. or less by the water manometer. In each instance the kink formed at the junction of the superior with the descending limb. In 1 of these cases the air, after passing the kink at 20 cm. pressure, found further obstruction at the point of crossing of the root of the mesentery. In 4 other instances obstruction occurred not in the duodenum but at the pylorus, which was tightly closed and required a pressure of from 15 to 25 cm. to overcome it. After passing the pylorus no further obstruction was encountered.

The conditions existing in all such postmortem experiments must of necessity be so entirely different from those which obtain during life, however, as to make it very doubtful indeed if the results so obtained can safely be applied to the explanation of the phenomena of acute dilatation.

More significant, I think, are certain animal experiments made by Kelling and verified by Braun. They found that in dogs upon which a gastrostomy had been done, and which had had time to recover from the effects of the operation, it was impossible to produce a condition of acute dilatation by inflating the stomach, for whenever a certain degree of distention was reached a vomiting reflex was excited and the stomach was emptied by vomiting and eructation. When, however, the animal was narcotized the stomach could be distended to the point of bursting without the least escape of air through the œsophagus and without any reflex attempt at regurgitation or vomiting.

That the development of acute dilatation of the stomach may be favored by anatomical peculiarities which lead to obstruction both at the cardia and in the duodenum seems altogether likely, but that such double obstruction is *necessary* to its production (as Kelling believes) seems to be clearly negatived by the fact that in many instances of typical and extreme dilatation vomiting and eructation have been present throughout the whole course of the disease.

While there is good reason then to doubt that obstruction at the cardia, or failure of the vomiting reflex, is essential to the development of acute dilatation, there is much evidence to show that obstruction below the stomach is present in very many of the cases and that it plays a role of considerable importance in the production of such dilatation.

Among the 69 autopsy reports studied an associated dilatation of the duodenum was noted 38 times (55 per cent.), and of these 38 cases 25 showed some form of obstruction below this dilatation (see Morbid Anatomy). In some of the remaining 13 cases the records are very incomplete and unsatisfactory, and it is impossible to say whether obstruction existed or not. But even if none of these cases is included, and if no allowance is made for the fact that occlusion by kink or by pressure might very easily be overlooked at the postmortem table, there still remains the fact that in 36 per cent. of all the autopsies definite obstruction was found in some part of the duodenum.

Further evidence of the frequency and importance of obstruction below the stomach is to be found, it seems to me, in the fact that the quantity of fluid vomited or removed from the stomach has been, in most cases, so enormous, and that this excessive amount of fluid can be most satisfactorily explained (as Müller has shown) by the assumption of some obstruction high up in the intestines by which the usual reabsorption of the (normally) large amount of fluid secreted by the stomach, pancreas, and liver is prevented. An analysis of the 25 cases in which some form of duodenal occlusion was shown to exist gives the following results: In 19 instances the dilatation ended at the point where the duodenum is crossed by the root of the mesentery, and the obstruction was ascribed to constriction by the tense mesentery. In 2 other cases (Meyer, Schultz) the dilatation extended to about the same point, but the obstruction was believed to be due to pressure of the displaced pyloric portion of the stomach. Twice (Buzzard, Petit) the occlusion was caused by a sharp kink at the duodenojejunal junction; once by a kink at the junction of the superior with the descending limb of the duodenum (Kausch), and once by an extravasation of blood (of unknown cause) between the mucous and muscular layers of the wall of the descending portion of the duodenum (Balster).

MESENTERIC OBSTRUCTION OF THE DUODENUM. The frequency with which mesenteric compression of the duodenum has been found associated with acute dilatation of the stomach and duodenum demands that this remarkable condition shall now be discussed in some detail. I have collected 18 fatal cases from the literature (Albrecht (2), Baümler, Binswanger, Kausch, Kelynack, Kundrat (3), Lehmann, Müller (2), Philippe, Robinson, Schmorl (Kelling), Schnitzler, Stieda, Walzberg).[3]

In addition to these fatal cases there are, among the cases of acute gastric dilatation which recovered, several (Bäumler, Müller, Robson, Schnitzler, Walzberg) in which the clinical picture was that of mesenteric obstruction of the duodenum, and in which a cure was effected by treatment (chiefly postural) directed toward the relief of the supposed mesenteric compression.

In view of the presence of bile and a diastatic ferment in the stomach contents and of the result of postural treatment in Baümler's second case it can hardly be doubted that the diagnosis of mesenteric obstruction was correct. The other cases referred to are scarcely less convincing.

In looking over the cases of mesenteric obstruction cited it will be seen that the condition, in its etiology, symptoms, duration; and prognosis, does not differ from that of acute gastric dilatation in general. Even the characteristic symptom of bilious vomiting has been present in most cases of acute dilatation of the stomach in which no mesenteric compression was noted.

Among the 69 fatal cases examined mesenteric obstruction was thought to exist 19 times (27.5 per cent.). In 19 other cases, as has been said, dilatation of the duodenum was found at autopsy, and, although in some of these instances other causes for the dilatation were discovered, in a number of them it seems very probable that mesenteric compression may have been present. If to these are added the several cases with recovery in which there is good reason to believe that the same condition existed, it becomes evident that obstruction of the duodenum by the mesentery must be regarded as a factor in the development of a large proportion (perhaps from one-third to one-half) of all the cases of acute gastrectasis.

The subject of the production of this form of duodenal incarceration has been carefully studied by Albrecht, who points out that the terminal portion of the duodenum, where it passes behind the root of the mesentery and lies upon the vertebral column and aorta, has under normal conditions not a round but a distinctly flattened circumference, owing to the pressure of the overlying mesentery and its contained superior mesenteric artery. This normal constriction can readily be seen in a cadaver in the dorsal position, and if a finger is passed into the duodenum and at the same time gentle traction downward is made upon the mesentery, the constriction becomes more distinct and is very evident to the examining finger. If now traction upon the mesentery is made in the direction of the axis of the pelvis—that is, downward and backward—the constriction may be converted into complete occlusion. With the small intestine lying altogether or chiefly in the pelvis, traction would be made upon the mesentery in just the direction needed to produce such occlusion. The presence of the small intestine in the true pelvis seems then to be a *sine qua non* to the production of such mesenteric occlusion of the duodenum, and this view is borne out by the autopsy findings of the recorded cases. In order that the intestine may make its way into the pelvis and there exert traction upon its mesentery, it is necessary that it contain little or no gas and that the mesentery be of suitable length—long enough to allow the intestine to hang in the pelvis without being supported by the pelvic floor.

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[3] A brief synopsis of these cases will be found in the Trans. Assoc. Amer. Physic., 1906, xxi, 601.

It is possible to demonstrate on the cadaver, as Albrecht and others have done, that traction on the mesentery in the direction of the axis of the pelvis may produce obstruction in the duodenum which will be impervious to water under considerable pressure. Box and Wallace, however, conclude from their experiments that the great distention of the stomach and the obstruction in the duodenum that can be produced in cadavers, in the dorsal position, by water pressure, depend not upon constriction by the root of the mesentery, but rather upon the direct pressure of the heavy stomach upon the duodenum where it crosses the lumbar vertebræ.

This question I have attempted to study in a series of 10 adult subjects. With the bodies in the dorsal position and the organs in place, the small intestine was removed and the mesentery gathered up into a mass and fastened to a cord which was passed into the pelvis and out through the anus and was allowed to hang free. To this different weights could be attached. The stomach and duodenum were then filled through the œsophagus, by a small tube of water from the tap, and the pressure necessary to overcome the duodenal obstruction estimated by connecting the tube with a mercury manometer.

The results differed greatly in the different cases. In 3 instances the obstruction seemed to be due almost entirely to the weight of the distended stomach upon the end of the duodenum, and with this raised very little interference with the flow of water into the jejunum was produced by a weight of 500 grams or even 1 kilogram attached to the mesentery. In each of these 3 cases a certain degree of gastroptosis existed. In the other cases a load of 500 grams (the approximate weight of the empty small intestines) produced an obstruction, to overcome which required a water pressure varying from 10 to 48 mm. of mercury. In some of these cases the stomach gradually distended to a point where it lay upon the duodenum, and by its weight increased somewhat the obstruction caused by the tense mesentery. In other instances the dilated stomach did not rest upon the duodenum and exerted no influence whatever upon the mesenteric obstruction.

The conclusion seems justified that in a certain proportion of normal individuals a pull upon the mesentery approximating, in direction and force, that which might be exerted by the empty small intestines hanging in the true pelvis can produce obstruction at the lower end of the duodenum which will require very considerable force to overcome.

The conditions essential to or which favor mesenteric obstruction may for convenience be divided into two groups: those which aid in the descent of the intestine into the pelvis, and those which favor the more complete occlusion of the duodenum.

For the entrance of the intestine into the pelvis three things, at least, seem necessary: dorsal decubitus, an intestine nearly or quite empty of gas and feces, and a mesentery of suitable length—long enough to permit the intestines to slip into the pelvis, and yet not so long that they may be supported by the pelvic floor.

It is possible that the frequency of such obstruction after operation (in 8 of the 19 cases) may depend in part upon the fact that at such times the intestines have been kept empty by fasting, purges, enemas, etc.

Other conditions which seem to favor the descent of the gut into the pelvis are chiefly those which disturb the normal equilibrium of the intra-abdominal pressure. Kundrat believed that a lax, flabby abdominal wall acted so by failing to hold the intestines in their usual position. Lehmann, in his case, which developed in the course of a double lower-lobe pneumonia, suggests that the weight of the hepatized lungs and the violent coughing efforts may have aided in forcing the small intestine into the pelvis. In the remarkable case of Schmorl the symptoms followed immediately upon a violent paroxysm of laughing. In one of Kundrat's cases they suddenly appeared in a typhoid-fever convalescent who had left his bed for the first time.

The duodenum is rendered more liable to serious compression, Schnitzler believes, by any unusual prominence (lordosis) of the lumbar vertebræ, or by an abnormally low position of the last portion of the duodenum, since the vertebræ become increasingly prominent as they descend.

Increased lordosis existed in 2 of the recorded cases (Kausch, Robinson), as well as in several other instances of acute gastric dilatation in which no mesenteric obstruction was recognized.

The same result was brought about in Philippe's case by a mass of clotted blood which lay behind the terminal portion of the duodenum and pushed it forward. He also demonstrated experimentally that by the injection of a quantity of tallow behind the lower part of the duodenum complete arteriomesenteric obstruction could be produced even with the small intestine not in the pelvis.

The line at which the third portion of the duodenum crosses the lumbar vertebræ varies much. Fawcett and Blachford, in studying this point recently, found the position of the lower border of the duodenum to vary between the upper border of the second lumbar vertebra and the disk between the fifth lumbar and the sacrum. Most commonly it crossed the third lumbar vertebra. In general the point of crossing was lower in females than in males. In view of the fact that the anterior curve of the lumbar vertebræ reaches its maximum at the fourth vertebra, it is obvious that a duodenum which lay upon the fourth vertebra would be more liable to serious compression by the mesentery than would one which crossed, say, the second vertebra. There has been some differences of opinion as to whether the constriction of the duodenum is caused by pressure of the whole root of the mesentery or chiefly only by the superior mesenteric artery which runs in it. The frequent use of the term "arteriomesenteric obstruction" indicates that the artery has often been looked upon as playing an important role in the production of the obstruction, and there can be no doubt that to the examining finger in the duodenum, with traction upon the mesentery, the most resistant portion of the constricting band is the tense, cord-like strand which represents the artery; but, on the other hand, in Baümler's fatal case the band of hyperemia and necrosis in the duodenum corresponded in width and position to the whole root of the mesentery and not to the artery alone.

From a consideration of the conditions just enumerated it is not difficult to see how a transient occlusion of the duodenum might occasionally occur; but why such an occlusion should become a permanent, unyielding obstruction, with its train of violent symptoms and usually fatal outcome, is, as Albrecht has pointed out, by no means easy to explain. It seems very unlikely that such a pinching of the duodenum should always result in complete and permanent incarceration. It is more natural to suppose that it sometimes produces only a temporary occlusion which passes unrecognized. What is it then that occasionally renders such an obstruction permanent and insurmountable?

The almost constant presence, in the fatal cases, of an enormously dilated stomach has led to the very general belief that this is the factor of chief importance in the production of a permanent incarceration. By the rapid increase in the size of this organ the intestine would tend to be crowded more and more into the pelvis, and, once there, would be prevented from escaping. At the same time the weight of the heavy stomach upon the mesentery would be likely to render futile every effort on the part of the duodenal and gastric musculature to overcome the obstruction.

While there is general agreement that the greatly enlarged stomach is of much importance in rendering the incarceration more complete and permanent, there is still a sharp difference of opinion as to whether the dilatation of the stomach or the obstruction of the duodenum be the primary act in the vicious circle by which the dilated stomach increases the duodenal obstruction, and this latter in turn augments the dilatation of the duodenum and the stomach.

Von Herff, Stieda, and Neck believe the dilatation of the stomach to be always primary and the mesenteric constriction to be purely a secondary happening. On the other hand, Müller and Lehmann regard the duodenal obstruction as of prime importance and the first act in the chain of events.

That this latter view is correct, for some cases at least, seems altogether probable from the fact that in 2 of the cases of mesenteric obstruction (Kundrat, Case II; Philippe) no mention whatever is made in the autopsy reports of any dilatation of the stomach. Also in such cases as that of Schmorl, in which the symptoms followed directly upon a violent paroxysm of laughing, it seems probable that the trouble began with the sudden constriction of the duodenum by the mesentery.

On the other hand, it is easy to see how a considerable dilatation of the stomach, either acute or chronic, might favor the production of mesenteric obstruction, and, when this has occurred, prevent its spontaneous recovery. Albrecht believes that in many of the instances of apparently acute gastrectasis there has been a preexisting chronic dilatation which has converted a transient mesenteric constriction of the duodenum into a permanent and complete obstruction, and that this, in turn, has brought about a sudden increase in the gastric dilatation with its train of alarming symptoms. Unfortunately for this theory there is little evidence in either the clinical or the autopsy records to support the idea that chronic gastric dilatation is a frequent antecedent of mesenteric obstruction.

*Chronic Mesenteric Obstruction of the Duodenum.* Kundrat and Glénard held that a persistent, incomplete obstruction of the duodenum by the root of the mesentery was not uncommon and led to a gradual dilatation of the duodenum and stomach. Albrecht cites 2 cases which he thinks may have been instances of this condition, and Robinson asserts that in the course of several hundred postmortem examinations he has met with fifteen or twenty examples of gastroduodenal dilatation due to such incomplete obstruction.

Nothing is known of the clinical manifestations of such chronic mesenteric obstruction, and, in view of its possible importance, it is very desirable that the attention of surgeons be directed to the need of careful examination of the lower part of the duodenum and the mesenteric root in operations affecting that part of the abdomen. It is quite possible that some of the instances of duodenal dilatation, to the frequency of which operators in this country

(Finney, Mayo, Ochsner) have recently called attention, may be due to such chronic mesenteric obstruction.

DIAGNOSIS. It is interesting, in looking over the records of the reported cases of acute gastric dilatation, to note that whereas the first case seen by most of the writers has passed unrecognized, subsequent cases observed by the same men have almost always been correctly diagnosticated during life. In other words, the condition is usually not difficult of recognition if the possibility of its occurrence be but held in mind. Intestinal obstruction and perforative peritonitis have been the conditions with which it has most frequently been confused. Once at operation the hugely dilated stomach has been mistaken for and treated as a pancreatic cyst. In some of the postoperative cases the persistent vomiting has been looked upon as only an exaggerated form of the usual postnarcotic vomiting.

The sudden onset of severe and continued vomiting, and especially the vomiting of very large amounts of bilious, non-fecal, fluid, should always suggest the possibility of acute gastric dilatation. A normal or subnormal temperature, rapid emaciation and collapse, obstinate constipation, and abdominal pain, tenderness and distention are other very common symptoms; but it must be emphasized that any one, or most, of these may be lacking in a given case. In two of the recorded cases vomiting has been absent throughout. In a few instances there has been neither pain, tenderness, nor distention. Occasionally diarrhœa has replaced constipation. The diagnosis is made definitely by the demonstration of the greatly dilated stomach by means of careful physical examination and especially by the use of the stomach tube. The existence of mesenteric obstruction of the duodenum is made probable by the demonstration, in the contents of the dilated stomach, of bile and of pancreatic juice, by the absence of distinctly fecal vomiting, and by the satisfactory results of postural treatment. It is possible that an examination of the pelvis through the rectum or vagina might show there the mass of collapsed intestine. Kelling suggests, as a means of determining whether or not complete obstruction exists at the lower end of the duodenum, the use of a high injection of a large quantity of concentrated (6 per cent.) salt solution. The usual effect of this is to empty the small intestine as well as the colon, and the presence or absence of fresh bile in the returned fluid would determine the question as to whether or not the duodenum was pervious.

TREATMENT. The effectiveness of treatment in acute dilatation of the stomach will depend, in large measure, upon the time at which it is begun. When the stomach has become dilated to its maximum and the patient has reached a state of extreme prostration and collapse, treatment has often been unsatisfactory and unavailing. On the other hand, when the disease has been recognized before this terminal stage was reached the results of treatment, in most cases, have been very gratifying.

The first step in treatment, whatever the cause of the dilatation may be, is the prompt and complete emptying of the stomach by means of the stomach tube. Owing to the often enormous size of the organ and the flaccid state of its walls, this is not always a simple task. The tube must be made to reach the most distant and dependent parts of the stomach, and changes in the patient's posture may be necessary in order that all the contents may be reached. This emptying of the stomach should be repeated at regular intervals, usually several times in the twenty-four hours. Food or drink by mouth should be absolutely interdicted. The excessive loss of fluid should be met by large saline enemas or by hypodermoclysis. Nutrition should be maintained as well as possible by rectal feeding.

In a considerable number of the cases the above-mentioned treatment has been all that was needed to effect relief of the symptoms and gradual recovery, but in view of the fact that some form of duodenal obstruction has been shown to exist in 36 per cent. of the fatal cases, and has probably been present in many others, the likelihood of such obstruction has to be considered in every case. If such obstruction is due only to a duodenal kink or to the weight of the dilated and overloaded stomach, the mere emptying of the stomach and the keeping of it empty may suffice to relieve the obstruction. It has been shown, however, that three-fourths of all the cases of duodenal obstruction have been caused by constriction by the root of the mesentery, and for the relief of such obstruction something more than the emptying of the stomach is needed. Schnitzler was the first to treat successfully a case of acute gastric dilatation on the assumption that the symptoms were due to mesenteric obstruction. The patient was changed from the dorsal to the belly position and all the symptoms promptly ceased. After several hours he was able to resume the dorsal position. The abdominal distention had disappeared, vomiting did not return, feces and flatus passed freely, and the patient made a good recovery.

In Baümler's case (already cited) the patient was kept in the knee-elbow position for a quarter of an hour in each two hours, the rest of the time lying on his belly. The symptoms disappeared promptly and the patient recovered.

The experience of Müller (Case IV) was even more striking. In this patient vomiting persisted in spite of lavage and elevation of the pelvis. It ceased immediately, however, when the belly position was taken. After two hours a dorsal posture was tried, but vomiting began again at once. The patient lay on her belly through the night and slept well. Next day she was able to take and retain food in this position, but every attempt to lie on her back was followed by vomiting. On the following day, however, she could lie on her back comfortably and thereafter had no further trouble.

Walzberg records a very similar experience (Case II). Lavage having been of no avail, the patient was turned upon his right side and three hours later upon his left side. Vomiting ceased immediately. Two days later, on attempting to lie upon his back, nausea returned and the lateral position had to be resumed. The patient made a good recovery. In one of Robson's patients (Case IV) the symptoms were relieved by keeping her in the belly position.

These results are so striking that is seems but fair to give every case of acute dilatation of the stomach the benefit of the doubt, and to try the effect of such postural treatment as seems most likely to displace the intestine from a position in the pelvis and to relieve a possible mesenteric constriction of the duodenum.

It should be noted, however, that in 1 case (Borchardt) the symptoms seemed to be made worse by the use of the belly position.

The relief of acute gastric dilatation by operation has been attempted a number of times. In 7 instances (W. H. Brown, Box and Wallace, Appel, Hoffmann, Jessop, Kirch, Wright) the stomach was opened and evacuated, but none of the patients recovered. Gastroenterostomy has been performed twice (Kehr, Körte), and both times unsuccessfully. Petit found at operation a sharp kink at the duodenojejunal junction. This he relieved by lifting up the jejunum and stitching it to the transverse mesocolon. The symptoms disappeared and the patient recovered. The advisability of a gastroenterostomy is open to serious question. There is quite general agreement that it should be employed, if at all, only as a last resort. In most cases it is unnecessary, and in those patients who have resisted all other methods of treatment it is doubtful, Borchardt thinks, if any good can be accomplished by operative measures.

SUMMARY. Acute dilatation of the stomach is a disease of considerable clinical importance and is deserving of wider recognition. It develops under a great variety of conditions, but is especially frequent after operations with general anesthesia. In a majority of the cases there has been an associated dilatation of the duodenum.

The mechanism of acute gastric dilatation is not yet clearly understood, but in a large proportion of cases (perhaps one-half) the dilatation is associated with, and seems to result from, some form of obstruction in the duodenum.

Much the commonest cause of such duodenal obstruction is the constriction of the lower end of the duodenum between the root of the mesentery, which crosses in front of it, and the vertebral column. Such constriction can occur only when the mesenteric root, with the superior mesenteric artery which runs in it, is stretched tightly across the duodenum; and this is possible, apparently, only when traction is exerted by the small intestine hanging over the brim of the pelvis. The development of mesenteric constriction is favored, therefore, by whatever facilitates the entrance of the small intestine into the true pelvis (dorsal decubitus, a mesentery of suitable length, an empty and collapsed state of the gut, etc.), and whatever renders the duodenum more liable to compression (increased lordosis of the lumbar vertebræ, low position of the duodenum, etc.). Great dilatation of the stomach would doubtless increase the liability to mesenteric obstruction by crowding the intestines into the pelvis, and would tend, by preventing the escape of the gut from the pelvis, to render such obstruction, when once formed, more complete and permanent.

Whether the dilatation of the stomach or the mesenteric obstruction of the duodenum constitutes, usually, the first step in the vicious circle by which both conditions are made worse is still uncertain, but there is little doubt that in some cases, at least, the duodenal obstruction is primary.

The symptoms and physical signs of acute gastric dilatation are usually so distinct that the diagnosis should not be difficult if the possibility of such occurrence be but remembered.

In spite of the high mortality (72 per cent.) among the reported cases, there is much evidence to show that the disease can usually be controlled and cured if the diagnosis be made and suitable treatment begun before complete collapse of the patient has occurred.

The following list of 82 cases,[3] in addition to the 2 cases reported herewith and the 18 cases of mesenteric obstruction already cited, constitutes the material upon which the present study is based:

Albu, Appel, Balster, Baümler, Bennett, Bettmann, Boas, Borchardt, Box and Wallace (3 cases), Braun (5 cases), Broadbent, H. H. Brown, W. H. Brown, Buzzard (Perry and Shaw), Dickinson, Dyson, Edmunds, Erdmann, Fagge (2 cases), Fenger, Fisher, Fränkel (2 cases), Goodhart (4 cases), Heine (2 cases), v. Herff (3 cases), Herrick (2 cases), Hoffmann, Hood, Hunter, Jessop, Kausch, Kehr (2 cases), Kelling, Kirch, Körte, Lieutaud, MacEvitt, Mahomed, Meyer, Miller and Humby, Morris (2 cases), Müller (2 cases), Petit, Riedel (3 cases), Robson and Moynihan (4 cases), Rosenheim, Schnitzler, Schultz, Simon, Thomson (5 cases), Valsalva, Walzberg, Willett, Wright (2 cases).

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[3] A report by Neck (Münch. med. Woch., 1906, p. 1561) of 4 additional cases of acute gastric dilatation appeared too late for inclusion in this paper.

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# THE OPERATIVE TREATMENT OF FRACTURES, ESPECIALLY OF THE LONG BONES.

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When a surgeon is called upon nowadays to treat a patient with a fractured femur, for example, the problem which immediately confronts him is whether to operate or not to operate. There was a time long gone by when the question of operation in such cases never arose unless it was a question of amputation or resection; operation for the purpose of putting the broken fragments in proper relationship with one another was not considered, since the proper adjustment of the fragments was thought to be done by extension, counterextension, and manipulation. The Röntgen rays have shown that most fractures of long bones set in this way have resulted in shortening due to overlapping of the fragments.

The question may be asked, Why should we operate on fractures which are almost certain to get well without operation? The answer is, For several reasons: (1) To prevent deformity of a limb, especially shortening, which is of the greatest importance in the lower limbs; (2) to prevent loss or impairment of function, as of pronation or supination; (3) to obviate delayed union or ununited fracture; (4) to give opportunity to repair the soft tissues—nerves, muscles, and bloodvessels; and (5) because, as a rule, it is the only way to get a perfect result. It is now generally agreed that for a fractured patella or olecranon the operative is the best treatment.

I believe that perfect adjustment in fractures of the long bones is seldom accomplished without operation, except in green-stick fractures and some transverse fractures in which the fragments may be only partially displaced and have not overridden, as in some Colles fractures. It is also often impossible to ascertain, after a supposed reduction, whether or not apposition has been obtained. In the case of a broken femur surrounded by muscles from two to four inches in thickness, who by palpation can tell anything about the relations of the fragments? We resort to measurement: a mistake of one-half to three-fourths of an inch may easily be made on